COST IPLANTA CA15223

WG1 Meeting:

"Comparing siRNA and miRNA technology and role for improving perennial plants"

Linked to "ISHS International conference on Grape Biotechnology", Bordeaux (FR),

July 17-18, 2018.

PROGRAM

*Grosshans H, **Filipowicz W**.Nature. **2008***

iPLANTA WG1 Meeting:

"Comparing siRNA and miRNA technology and role for improving perennial plants"

INRA – VILLENAVE D'ORNON - BORDEAUX

The WG1 meeting will focus siRNA and miRNA technology and role in regulating different processes in plants and in their interaction with other organisms. A focus will be given in determining which technology has been favourably used for annual and perennial plant species including the rootstock-to-scion transfer of the silencing virus resistance signal. A scientific dissemination plan, including the preparation of a review on RNAi technology, will be discussed.

The meeting will be organized with the following working program:

• July 17 (Morning): registered WG1 experts will attend the "ISHS international conference on Grape Biotechnology", at ENSEIRB, amphi Matmeca, 1 Avenue du Docteur Albert Schweitzer, 33402 Talence

• July 17 (Afternoon): iPLANTA WG1 meeting, registration at 2:00 pm, starting of the meeting 3:00 pm, at INRA- Bordeaux, 71Avenue Bourlaux, 33140 Villenave d'ornon.

• July 18 (morning): WG1 scientific session at INRA- Bordeaux, 71Avenue Bourlaux, 33140 Villenave d'ornon with selected oral presentations on major advances on RNAi technologies.

• July 18 (afternoon): WG1 discussion and planning of WG1 scientific and dissemination activities at INRA- Bordeaux, 71 Avenue Bourlaux, 33140 Villenave d'ornon .

New specific topics will be introduced by identifying and invite 1 or 2 international speakers. All presentations will be oral. As for all iPLANTA meetings will be reimbursed only experts selected for abstract presentation as reported in the following program.

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IN COLLABORATION WITH:

NATURAL RESISTANCE OF THE DIPLOID MUSA BALBISIANA PISANG KLUTUK WULUNG (PKW) BANANA PLANT TO INFECTIOUS ENDOGENOUS BANANA STREAK VIRUS SEQUENCES IS DRIVEN BY TRANSCRIPTIONAL GENE SILENCING.

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Keywords: Banana, endogenous banana streak virus (eBSV), methylation, epigenetic

The genome of banana (Musa sp.) harbours multiple integrations of Banana streak virus (eBSV), whereas this badnavirus does not require integration for the replication of its ds DNA genome. Some endogenous BSV sequences (eBSV), only existing in the Musa balbisiana genome, are infectious by releasing a functional viral genome following stresses such as those existing in in vitro culture and interspecific crosses context. The structure of these eBSV is much longer than a single BSV genome, composed of viral fragments duplicated and more or less extensively rearranged.
Wild M. balbisiana diploid genotypes (BB) such as Pisang Klutuk Wulung (PKW) harbour such infectious eBSV belonging to three widespread species of BSV (Goldfinger - BSGFV, Imové – BSIMV and Obino l'Ewai - BSOLV) but are nevertheless resistant to any multiplication of BSV without any visible virus particles. Using deep sequencing of total siRNAs of PKW we underlined the presence of virus-derived small RNA (vsRNA) from eBSOLV, eBSGFV and eBSIMV by blasting sequences against the 3 BSV species genomes. Interestingly, we showed that hot and cold spots of vsRNA generation do not target similar viral sequences from one eBSV species to the other but are directly correlated with the structure of the integration. vsRNA are enriched in 24-nt class which represent about 75% of the total 21-24nt siRNA matching eBSV. We also demonstrated that eBSV are highly methylated in the three different sequence contexts (CG, CHH and CHG) throughout the whole sequence of eBSVs with no difference in methylation profile between siRNA producing and non producing areas. Interestingly, methylation patterns of all three eBSV are similar whereas they are located in different genomic context; eBSOLV being in a TE rich area whereas eBSIMV and eBSGFV are in genes rich region. It seems that eBSV are controlled mainly by epigenetic mechanisms similar to those described for transposable elements (TE). All together, our data indicate that eBSVs in PKW genome are likely silenced at the transcriptional level and this is probably responsible for the natural resistance of this genotype to the activation of such infectious eBSV as well as infection by external BSV particles.